decreases not only due to chemotherapy-induced neutropenia, but also due to the reduction of serum antibody titers gained from previous immunizations.Children are at an high risk for developing hepatitis B virus (HBV) infection from immunosuppression secondary to chemotherapy and multiple blood transfusions. Most of the children infected with HBV develop chronic hepatitis. An Indian study by Jacob Puliyel et al, showed at 6 years of age, protective levels of anti-HBs antibody (>10 mlU/mL) were present only in about 59% of those immunized. By 11 years, only 13% had protective levels. The increasing potential for the cure of childhood ALL emphasizes the need for a method of reducing hepatitis and its sequelae in these children. Our study revealed only 68.8% children with newly diagnosed ALL had protective antiHBstitres (>10IU/L), while 31.2% children had no immunity tohepatitis B despite presumed vaccination as part of the UIP schedule.

**Conclusion**: A significant number of newly diagnosed children with ALL lack protective anti HBs titres despite being vaccinated according to Universal Immunization Programme and Combined passive active immunisation should be considered for them.

### LM-1_V1.3
### CLINICAL PROFILE AND OUTCOME OF EARLY T-PRECURSOR (ETP) ACUTE LYMPHOBLASTIC LEUKEMIA (ALL) IN A TERTIARY CARE CANCER CENTRE

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**Introduction**: ETPALL is a recently well described high-risk subset of T-ALL with early differentiation arrest and showing features overlapping with AML and with poor prognosis which can potentially be improved with intensified induction using dexamethasone and high-dose L-asparaginase. We audited our experience with ETP-ALL for its clinical profile and outcome.

**Methods**: We retrospectively evaluated all children (<15yrs) diagnosed with ETP-ALL or Near ETP-ALL from January 2012 to December 2015. All were treated with institutional ALL protocol (modified MCP-841) with 4drug induction incorporating prednisolone and high dose Ara-C in consolidation (16g/$m^2$). From June 2014, the protocol was amended by substituting prednisolone with dexamethasone (10mg/$m^2$/dy) in induction along with higher intensity of L-asparaginase (10000 U/$m^2$/dose).

**Results**: T-ALL was diagnosed in 283 children, of whom 34 were ETP-ALL (14%) and 5 were Near ETP-ALL (1.7%): Median age was -13 years; Male : female- 2.5:1; Bulky disease-18%; mediastinal mass-12.8%; median WBC count- 10.1x$10^9$/$mm^3$; Hyperleucocytosis (>100x$10^9$$mm^3$)- 10%; No CNS involvement. Demographics of T-ALL in same period: median age- 9yrs; male: Female 4:1; median WBC count- 50x$10^9$ /$mm^3$; hyperleucocytosis-36.7%.
Of 39 children with ETP-ALL, 32 took treatment. At a median follow-up of 10 months (range 3 - 50months), the 2-year OS is 52.5% and EFS is 53.7%. Median OS is not reached as yet. Of 32 patients, 15 received prednisolone based induction and 17 received dexamethasone based intensive induction. Overall 8 (25%) died during induction therapy (dexamethasone arm – 29.4%, prednisolone arm -20%). Of the evaluable 24 patients (dexamethasone arm-12, prednisolone arm-12),18 patients (75%) achieved complete morphological remission at the end of induction (dexamethasone arm-58%, prednisolone arm-92%, p=0.15). Of 14 patients whose Minimal Residual Disease (MRD) data were available (12 of whom received dexamethasone based induction), 5 patients were MRD negative post induction (35.7%), and corresponding morphological remission was seen in 9 (50%). Of the 9 patients who were MRD positive post induction,7 (78%) cleared MRD post subsequent cycles .There has been no relapse so far and the projected 2-year DFS is 89%.

**Conclusions**: ETP-ALL constituted 16% of T-ALL children. Children with ETP-ALL had higher median age, low disease burden at presentation and infrequent CNS involvement. The disease free survival with this protocol using high dose cytarabine based consolidation is good. However intensification of therapy in induction did not improve remission rates but led to higher induction mortality and poorer overall outcome in our low-middle income setting.

### LM-1_V1.4
### TOXICITY PROFILE OF L-ASPARAGINASE IN CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKAEMIA – SINGLE CENTRE EXPERIENCE

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**Introduction**: Acute lymphoblastic leukemia (ALL) is a hematologic malignancy that predominantly occurs in children between 2 and 10 years of age. L-asparaginase is an integral component of treatment for patients with ALL and since its introduction into pediatric treatment protocols in the 1960s, survival rates in children have progressively risen to nearly 90%. The effects of L-asparaginase on hemostasis during induction chemotherapy are less defined in children. These hemostatic modifications induced by L-asparaginase are relatively rare and mild in children, but their incidence has not yet been well evaluated and their Toxicity profile in Indian children is very rarely documented.

**Aims and Objectives**: Primary Objective: To demonstrate the toxicity profile of L- Asparaginase in children with Acute Lymphoblastic Leukaemia specifically to evaluate hyperlipidemia and changes in coagulation and thrombotic risk.

**Secondary Objective**: To study the differences in toxicities of L-Asparaginase between the two types of L-Asparaginase (E coli and Pegylated).

**Methodology**:

- Children after the diagnosis with acute lymphoblastic leukemia underwent baseline investigations with Liver function tests, Lipase, PT, PTT, INR, Random blood sugar and Lipid profile prior to the onset of treatment and thereafter weekly during induction and re induction periods.
- Antithrombin III and fibrinogen were done twice during induction (day 15 and 29) and once during reinduction therapy (on day 22).
- Demographic data, clinical details, details of diagnosis, Laboratory values were recorded in a proforma. Analyzed data were analysed using SPSS 16. The study was approved by our institution ethics committee.
- The study was funded by Tiara Hemophilia Cancer Foundation, NGO supporting in Chennai supports pediatric cancer researches.

**Results**: 80 cases were included in this study of which 53 were males and 27 were females. Pre B cell leukemia were 57 and T cell Leukemia were 23. Type of L- Asparagine used were E coli Asparaginase (n-50) and Pegylated L Asparaginase (n-30). Complications seen were elevated lipase level without any evidence of clinical pancreatitis 12.5%, Hyperglycemia 12.5%, hyperlipidemia 10%, Thrombosis 2.5%, Allergies 5.6%, elevated PT/PTT 10%; Low AT 3 (15.5%) and low fibrinogen (16.5%).
There is no Statistical difference between the two groups of L-Asparginase studied, however increase side effects were noted in Ecoli Asparaginase group.

**Conclusion**:

- Asparaginase is a critical component of all pediatric ALL protocols, with many protocols incorporating prolonged and high intensity L-Asparaginase treatment, it is important that our Indian data be available of all potential treatment-related toxicities.

### LM-1_V1.5
### HYPERGLYCEMIA DURING INDUCTION CHEMOTHERAPY OF PEDIATRIC ACUTE LYMPHOBLASTIC LEUKEMIA

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**Introduction**: Hyperglycemia occurs in about 10-16% of children during induction chemotherapy for acute lymphoblastic leukemia (ALL), which includes steroids and L asparaginase. Patients may present with diabetic ketoacidosis or non-ketotic hyperglycemic hyperosmolar syndrome. Hyperglycemia has also been reported to suppress immune function by inhibition of endogenous production of interleukins 2, 6 and 10 and uncontrolled diabetes is a risk factor for developing invasive fungal infections.

**Materials and Methods**: This study analysed the incidence and outcome of hyperglycemia in pediatric patients (0-14yrs) during induction